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Shock in Myocardial Infarction

In evaluating the effects of treatment on the natural prognosis of "shock" accompanying myocardial infarction, it is essential to define this type of circulatory failure regardless of the possible misuse of the term "shock" in this context. The generally accepted clinical pattern in all types of shock consists of slightly cyanotic pallor, cold skin, excessive sweating, restlessness, weakness, and tachycardia. These signs vary in occurrence and intensity. The apparent critical characteristic of the life-endangering shock of myocardial infarction is severe prolonged hypotension which may occur at the onset of the attack, during the first day, or from the second to the fourth day.

The reported fatality rates in untreated shock of myocardial infarction range from 60 to 90%. The variation arises largely from the differences in criteria of "severity" and "duration." The two elements may operate independently or concomitantly in affecting prognosis. For instance, a blood pressure that falls to an unobtainable level will represent a threat to life if it persists for 15 minutes or even less. A fall in systolic pressure to 85 mm. Hg. which lasts for 4 hours, if accompanied by such evidence of circulatory inadequacy as an hourly renal output of only 10 ml. of urine, is equally likely to lead to death. The preinfarction blood pressure apparently influences the critical level of shock production. Thus, a hypertensive patient with fixed pressures of over 180/110 mm. Hg., may enter a shocklike state when his pressure falls to 110/70 mm. Hg., whereas a person whose "normal" pressure is 94/60 mm. Hg. may exhibit no apparent shock with a fall to 80/50 mm. Hg. Fixing an arbitrary critical level of blood pressure or duration of hypotension which is applicable in all cases is impossible. Accumulated experience and reports in the literature, however, indicate that in a normotensive patient, a decrease in systolic blood pressure to below 85 mm. Hg which persists for over 1 hour is generally accompanied by the other features of shock and, if not treated, denotes a poor prognosis. If the hypotension persists over 3 hours, the shock becomes irreversible and 90% of such cases terminate fatally regardless of possible transient response to heroic therapy.

Approximately one-half of all patients exhibiting systolic hypotension of 60 to 80 mm. Hg. for one-half hour or less spontaneously recover without demonstrable ill effects. Hypotension with systolic blood pressure levels between 70 and 85 mm. Hg. may be maintained for 12 to 72 hours in certain patients who show no other manifestations of shock and who may excrete from 200 to 400 ml. of urine daily. However, over 80% of these patients, if not treated, develop severe and fatal irreversible shock. Attempts at correcting the hypotension are strongly recommended in such cases.

Current knowledge of the basic mechanism of the shock of myocardial infarction and irreversible shock dictates to some degree the therapy employed.

Two elements leading to hypotension are recognized as being precipitated by myocardial infarction. The first consists of myocardial failure and reduced cardiac output. The frequent occurrence of mild to severe pulmonary edema and of prolonged circulation rates, as well as ballooning of areas of heart muscle as observed by roentgenkymography, clinically corroborates this concept. In such cases, administration of oxygen by nasal cannula, mask, or tent seems warranted. Digitalis glycosides are widely recommended, but whereas no contraindication exists for their use in nontoxic doses, they appear to be of little or no immediate benefit except in atrial flutter or fibrillation. The second element leading to hypotension is a peripheral vascular reaction resulting in (a) failure of maintenance of arterial resistance in the presence of lowered cardiac output, and (b) possible decrease of peripheral and visceral venous tone with maintenance of constriction of outlet visceral veins resulting in venous pooling as demonstrated experimentally in other forms of hypotension by Smith and Hoobler and by Weil, et al.

Irreversible shock is likely to develop if hypotention is not corrected within 3 hours. Recovery occurs in only about 10% of such patients treated with pressor agents. The causes of irreversible shock are not known, but several reasons have been given for the failure of a patient to respond to treatment and for the transiency of pressor responses to drugs. These are (a) the release of ferritin or vasodepressor substance (VDM) by the hypoxic liver, (b) the development of acidosis which diminishes the effects of pressor amine drugs and incidentally tends toward precipitation of cardiac arrhythmias, (c) decreased blood coagulability and formation of multiple thrombi in the lungs, brain, and kidney, (d) absorption into the blood of rapidly developing bacterial exotoxin from the hypoxic intestinal tract, (e) further damage to the myocardium by ischemia and hypoxia as evidenced specifically by faulty carbohydrate metabolism of the muscle, and (f) possible adrenal secretory insufficiency. In the light of these possible causes, oxygen inhalation, administration of heparin and of adequate amounts of fluid and carbohydrate by parenteral or oral routes, use of intestinal antibiotics, e.g., Bacitracin or Polymyxin, and alkalinizing therapy without concomitant sodium retention may be considered theoretically valuable. Unfortunately, none of these measures has been clinically proved to increase the chances of a patient's recovery, although all have been effective in animal experimentation. In certain cases, administration of adrenal cortocoids, such as hydrocortisone 100 to 300 mg. intravenously has seemed to potentiate the action of the pressor amine drugs, as has been demonstrated experimentally; generally, however, such therapy is ineffective.

Other measures used in the treatment of shock include: (1) Rapid transfusion or plasma infusion (except in patients with elevated venous pressure) by the intravenous route (3 to 10 ml. per minute) or by the intra-arterial route (10 to 50 ml. per minute). (2) Hypothermia has been reported by Weil in shock of bacteremic infarction and by Vogelsang in shock

of myocardial infarction. The latter author reported that three patients with severe hypotension promptly recovered after infusion of 250 ml. of iced (4° C.) plasma. No confirmation of these results has been reported. (3) General care of the patient is important including adequate rest without excessive use of opiates which cause respiratory depression or antidiuretic effects, attention to bowels and skin, and oral intake of food. Any serious arrythmia should be corrected, but it should be noted that Pronestyl may accentuate the hypotension. (Sampson, J. J., Treatment of Shock in Myocardial Infarction: Dis. Chest, XXXIII: 667-671, June 1958)

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Significance of the Serum Amylase Determination

Since it has become well established that an elevated serum amylase level is the "sine qua non" of a valid diagnosis of acute pancreatitis, this laboratory procedure has been accepted as an essential part of the routine workup of the acute abdominal case. As data have been accumulated and analyzed, a considerable number of published reports have stressed that not only is the serum amylase elevated in many nonpancreatic conditions, but that a number of commonly employed drugs may significantly increase the amylytic activity of the serum. As a result, the clinician finds it increasingly difficult to interpret an abnormal serum amylase report and he is continually beset by the fear that too rigid adherence to "the elevated amylase = acute pancreatitis" concept may—in the acute abdominal case—lead to a serious diagnostic error.

With the hope that a survey of a large series of serum amylase determinations might eliminate some existing confusion and shed light on the significance of the serum amylase determination, the authors reviewed the results of all such studies carried out at Georgetown University Hospital from 1949 through 1956. During this 7-year period, the spectrophotometric technique of Smith and Roe was employed and the procedure was in each case personally carried out or supervised by the same technician. A range of 40 to 130 units was accepted as normal. Each determination was carefully correlated with the clinical record of the case including available surgical and postmortem findings. The reviewed material consisted of 1840 determinations carried out in 977 cases as indicated by table. The serum amylase was abnormally high in 494 patients and in 379 (76%) of these, the elevation was related to pancreatic disease. In 78 cases, an abnormally low amylase was found.

This review of a large number of serum amylase determinations demonstrated that, although a rise occurs consistently in acute pancreatitis, a similar elevation occurs with sufficient frequency in other nonpancreatic disease to limit the diagnostic value of this laboratory aid.

The concept that an elevation of the serum amylase is more importantly related to impairment of free egress of the pancreatic juice into the duodenum than to pancreatic trauma is supported by clinical evidence. Pancreatic calculi and sphincteritis are conditions in which obstruction to outflow of pancreatic juice is obvious, while after surgical resection of the pancreas which is associated with considerable trauma, hyperamylasemia is not common. It is not possible to relate the factor of stasis of pancreatic juice to the elevation of the amylase in many nonpancreatic conditions, such as peritonitis and cholecystitis, although in some, a remote relationship, i. e., increased intraluminal pressure in intestinal obstruction, is theoretically logical.

Studies to be reported from the authors experimental laboratory confirm the concept that obstruction of the pancreatic ducts and not necrosis of the pancreas is responsible for a rise in serum amylase. As a result of these investigations, evidence is accumulating that another enzyme, desoxyribonuclease (DNAase), is specifically related to cellular necrosis of the pancreas—a fact that may have significant clinical value.

Although the opposite view has been commonly held, the studies indicated that neither the height of the amylase elevation nor its duration served to distinguish acute pancreatitis from other acute abdominal conditions in which hyperamylasemia occurred. Furthermore, serum amylase values were not helpful in distinguishing between the edematous and the necrotic form of acute pancreatitis. In chronic pancreatitis, an elevated amylase was observed in less than half of the cases on admission, although an elevation was later identified during the period of hospitalization in additional cases with the result that in over two-thirds of cases a diagnostic elevation was noted. Hyperamylasemia is notably absent in cancer of the pancreas; in fact, a depression of the serum amylase level is more characteristic of this lesion.

Direct operative attack on the pancreas provoked a rise of the serum amylase in less than one-fourth of the cases, suggesting that as long as free drainage of the pancreatic ductal system is maintained, trauma in itself does not result in the elevation.

Repeated determinations of the serum amylase are desirable during the course of acute and chronic pancreatitis, not only as an index of the activity of the inflammatory process, but as was observed in a number of cases in this series, a protracted elevation more often than not indicates the development of a peripancreatic collection or pseudocyst.

In nonpancreatic diseases, an elevation of the serum amylase was observed in a sufficiently large number of cases to dispel the belief that a specific relation exists between pancreatic disease and hyperamylasemia. Undoubtedly, the rise in some of these conditions, i. e., epidemic parotitis, penetrating duodenal ulcer, is at least partly the result of secondary pancreatitis, but clearly, in many the pancreas was histologically normal.

The possible role of various drugs as an explanation of serum amylase elevation in these nonpancreatic disorders was continually kept in mind in this review. In fact, studies concerning the influence of morphine, codeine, Demerol, Prostigmine, and intravenous alcohol on the normal serum amylase level are now being completed. While these investigations clearly confirmed the fact that some of these agents provoke an elevation of the amylase, cases in which the drug factor was the possible explanation of the abnormal serum amylase level were excluded from this study.

The amylase level was elevated in every case of epidemic parotitis, in half of whom there was no associated abdominal pain. The fact that half the cases were complicated by mumps meningo-encephalitis is, in the authors' opinion, more indicative of the severity of the disease than of any specific relationship to the elevated amylase. It is interesting that in one of three cases of suppurative non-epidemic and unilateral parotitis, a rise in serum amylase was found.

Cholecystitis of both the acute and chronic type is attended by an abnormally high serum amylase level in 17% of cases. An equally significant incidence of hyperamylasemia was observed in calculous disease of the common duct, while in sphincteritis, the amylase was elevated in all of five cases. Whether the elevations of amylase associated with disease of the extrahepatic biliary tree in cases having no demonstrable pancreatic inflammation is due to the cholecystic or choledochal disease itself or the result of associated spasm of the sphincter of Oddi, is debatable.

A noteworthy incidence of hyperamylasemia was in association with duodenal ulcer (17%) and with gastrojejunal ulcer (66%). However, an elevated amylase rarely was observed in gastric ulcer. In almost one-half of the cases of posterior penetration of a duodenal ulcer, an elevation occurred, while in only two of 13 cases of free perforation was it observed. This latter incidence may be indicative of early institution of surgical closure of the perforation, although the authors observed no relation between the interval of time from perforation to surgical closure and hyperamylasemia. The high incidence (41%) of elevated amylase following partial gastric resection is undoubtedly related in part to factors that impair free drainage of pancreatic juice. A sufficiently high occurrence of hyperamylasemia was encountered in intestinal obstruction (20%), mesenteric thrombosis (33%), and peritonitis (69%) to establish the clinical fact that the demonstration of an elevated serum amylase in association with one of these conditions is not in itself an indication that pancreatitis coexists.

In advanced renal disease and in a large unrelated group of miscellaneous conditions, an occasional elevated serum amylase may occur. The finding of a distinctly subnormal amylase level or the absence of amylytic activity of the serum is not unusual. In the presence of pancreatic disease, such a depressed amylase level is indicative of either carcinoma or advanced chronic pancreatitis. Of the nonpancreatic conditions associated with

hypoamylasemia, cirrhosis and biliary tract disease are the most conspicuous. (Abruzzo, J. L., et al., Significance of the Serum Amylase Determination: Ann. Surg., 147: 921-927, June 1958)

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Treatment of Infectious Mononucleosis

A review of 1500 cases of infectious mononucleosis treated in the Department of Health and Preventive Medicine, Syracuse University, during the past 10 years confirms the therapeutic failures of chemicals, but reveals a specific regimen of treatment that will produce consistently satisfactory results. This article presents the elements of that regimen.

To establish a definite diagnosis in a disease as protean as infectious mononucleosis is not always easy. In many instances, its presence is suspected only after an unsuccessful trial of antibiotics, particularly if the services of a clinical laboratory are not utilized.

One should suspect that a patient with acute inflammation of the respiratory tract has infectious mononucleosis if (a) swollen lymph nodes are palpable outside the drainage area of the inflamed tissue, (b) the spleen is enlarged, (c) there is a prolonged febrile course with systemic manifestations, or (d) empiric antibiotic therapy fails to produce a response. Any bizarre group of symptoms and signs should arouse one's suspicion that the patient has mononucleosis.

A positive diagnosis of infectious mononucleosis is made in the clinical and serological laboratory. However, laboratory findings are not entirely predictable. The finding of a lymphocytosis with an increase of atypical lymphocytes to 20% or more usually establishes the diagnosis. A lesser number of atypical lymphocytes is observed during the extremes of mononucleosis, but this is also seen in a great variety of diseases. The authors believe that an increase of atypical lymphocytes to 20% or more is pathognomonic of the mononucleosis syndrome and that a positive heterophil agglutination is only confirmatory. Heterophilic agglutination—like the presence of atypical lymphocytes—is not specific for mononucleosis and frequently additional absorptive procedures are necessary to give it significance.

Not all patients who have infectious mononucleosis require treatment. The physician may discover the disease during a routine blood examination for a patient who is asymptomatic or has only vague or slight symptoms. Many such patients recover promptly without treatment. However, infectious mononucleosis usually produces signs and symptoms disabling enough to require medical supervision; it is in this group that a specific therapeutic regimen will produce results that are uniformly good.

The fundamental unit of therapy in infectious mononucleosis, therefore, is rest, based on the need for conservation of energy and the shift

of energy to tissues concerned with defense and repair. The amount and type of rest are calibrated to individual needs. Activity must be restricted until the disease process has abated. The return of the white blood cell count to normal is the best indicator of this end point.

Most patients with acute infectious mononucleosis have anorexia and painful deglutition. These symptoms can create problems in nutrition and, sometimes, relatively severe dehydration and electrolyte imbalance. Early in the acute phase, it is often necessary and wise to support the patient with intravenous sodium chloride, glucose, and water. This simple supportive measure alone often reduces the metabolic disturbance and places the supply and demand of energy in positive relation. If deglutition is painful, liquid or soft food is necessary. The diet should contain as many calories and as much protein as the patient can tolerate.

Misinformation, fear of the unknown, and the natural history of infectious mononucleosis lie heavily on the shoulders of the busy tense young patient. Education of the patient must begin as soon as the diagnosis is established. Relaxation and conservation of energy quickly follow understanding in most instances. The physician should avoid setting arbitrary limits on the period of disability and he should not initiate acceptance of the treatment program through fear of consequencss. Recognition of the patient's environmental stresses enable him to assist in minimizing their impact. As the acute phase of illness subsides, the supervising physician should start a positive program to return the patient to activity, beginning by planning the program with the patient and then gradually putting the plan into action. Patience and understanding are necessary ingredients of the program and it should emphasize positive activity (things that can be done) rather than negative activity (those that cannot).

Medical agents used in treatment of infectious mononucleosis during the past 20 years are voluminous. For the most part, they comprise four groups and are listed in a table: symptomatic agents, substances that influence (or theoretically influence) human metabolism, antibacterial agents, and steroid hormones. Review of the practical problems involved in mononucleosis explains the rational application of some of the substances in each group. In the present series, only the drugs in the first group—symptomatic agents—were used in all cases.

Several surgical complications are associated with mononucleosis. At times they are serious. Heading the list is a ruptured spleen. The framework of the spleen often becomes disorganized and friable in the presence of infectious mononucleosis. Therapy with reference to this organ should be prophylactic. Repeated attempts to palpate the spleen should be avoided. Spontaneous rupture is possible, and abdominal pain, nausea, vomiting, and shock should arouse one's suspicion that it has occurred. Prompt surgical intervention in either traumatic or spontaneous rupture of the spleen can be lifesaving.

Enlarged lymph glands in the abdominal area may simulate many surgical conditions. The clinical signs and symptoms may be classic of acute appendicitis. In the early stages of mononucleosis, the absence of a typical blood count may further complicate the picture. Even more distressing is an almost pathognomonic picture of acute appendicitis with a typical mononucleosis blood pattern. There is no clear-cut way of separating these two problems and yet separation is mandatory. The authors found that hourly clinical observation is the best aid and will lead to differentiation in about 75%. Unfortunately, 25% defy classification. In the latter cases, medical intelligence will dictate abdominal surgery.

Therapy designed to lessen the impact of the destructive and disorganizing phase of the inflammatory reactions involving the nervous system is experimental. Clinical improvement in specific cases of mononucleosis complicated by neuritis and paralysis reportedly has followed administration of 2, 3-dimercaptopropanol (BAL). Because of the relatively low incidence of this type of pathologic process in mononucleosis, statistically significant results of any therapy are probably unattainable.

Rapid reversal of paralysis of the seventh cranial nerve not associated with mononucleosis has been observed in several clinics following the administration of steroid hormones. This experience combined with the well-established resolution of the pathologic process of mononucleosis subsequent to steroid therapy would suggest that—at least experimentally—steroids should be administered to patients with mononucleosis who have involvement of nerve tissue. (Cronk, G.A., Naumann, D.E., Treatment of Infectious Mononucleosis - A Review of 1500 Cases: Postgrad. Med., 23: 605-611, June 1958)

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Tuberculosis - A Disease of Old Age

Since the beginning of the century and especially since the most recent advances in the battle against tuberculosis through effective chemotherapy, the age distribution of the disease has changed radically in those parts of the world where a concerted attack on tuberculosis has been possible. The first great change was accomplished through effective sanitation of the milk supply which resulted in practically complete control of bovine tuberculous infection in a few countries, notably the United States; the secondary manifestations of bovine infections—particularly tuberculous osteomyelitis—have since become comparatively rare.

This achievement in combination with the enlightened concept that exposure to the human bacillus in home life is the main source of clinical pulmonary tuberculosis resulted in a decline of the mortality among children to very low values even before the era of chemotherapy. American statistics

show a remarkable decrease from about 200 deaths in children per 100,000 population between 1900 and 1954. During the past decade it has almost reached the zero point.

The decline of the mortality curve for the total population during the past 50 years which has proceeded at an even pace with elevations only during the two world wars shows some of the factors which are at work in the tuberculosis problem when the data are analyzed as to sex, race, and age. In the United States, the picture is greatly influenced by the prevalence of the exudative and progressive forms of pulmonary tuberculosis among the nonwhite and immigrant elements of the population, chiefly the Negroes, the Puerto Ricans, and the refugees from war-torn countries, and to some extent also by the American Indian. Undoubtedly, the decline of the mortality curve as it appears in published statistics conveys an over optimistic impression if it is interpreted in terms of "cured" or "healed" tuberculosis. It is impossible to measure over a period of only a few decades the true killing effect of a chronic disease if that disease in individual patients can outlast the period of recording. It is known that the lower mortality is not the mere result of the decrease in the number of new active cases. Therefore, it must be assumed that, especially during the past 10 years of chemotherapy, the swiftness of the decline of the mortality—at least in part—is not so much due to the number of persons cured of tuberculosis as to the increasing chronicity of the disease which has shifted its weight into an older age group of the population. This change has become one of the most burning problems in tuberculosis care.

In the past, the cirrhotic, fibroid, and fibrocaseous forms of pulmonary tuberculosis have been a rather uniform finding in elderly patients, who usually had been tuberculous for many years, although with new bronchogenic exacerbations. In recent years, it has been a surprising and somewhat puzzling experience to find many fresh exudative involvements in patients where the absence of tuberculosis had been established when they were already in the old age group. The clinical-pathological picture of tuberculosis in the aged, therefore, differs less from that seen in young people than one should expect. There has been speculation that this type of senile disease might be due to the fact that first infection with the tubercle bacillus occurs increasingly at a higher age than used to be the case and that a shorter period is available during which subsequently an adequate acquired immune resistance can develop. However, this consideration can hardly apply to the present-day senile patient whose childhood still fell into the period when primary infection occurred in the great majority of children. The likeliest explanation is that in previous generations, when roentgenologic examination was either not available or not applied widely, when the public as well as the medical mind associated tuberculosis essentially with the young, the disease was not expected and consequently not looked for in old persons. Its symptoms—if any—and eventually even death in many cases were attributed to nontuberculous causes.

The question of the infectiousness of tuberculosis in some ways has become more complicated through modern chemotherapy rather than simplified. For practical considerations, the authors distinguish three main groups of sputum findings: in the first group, the sputum remains abundantly positive both on direct microscopic examination and on culture even under vigorous chemotherapy; in the second group, the production of bacilli is diminished to only occasional and scant positive results on culture; in the third group, the sputum is completely negative on all tests by both direct microscopic examination and culture. The patients belonging to the first group cannot be permitted to return home to live with children and young adults even under the most favorable living conditions. Those in the second group are considered by many authors as practically noninfectious and reports have been published indicating that they do not spread active disease and do not even lead to conversion of the response to tuberculin from negative to positive among persons living in their environment.

One may not believe in the danger of occasional exposure to infection as a major factor in the distribution of tuberculosis, but the continuous contact in intimate home life has long proved itself to be the essential cause of clinical disease. When the findings from routine roentgenologic examinations of the general population were compared with those in persons known to have lived in close contact with tuberculous individuals, it was found that the incidence of clinically important tuberculosis was three times as high in the latter group as among even the socially underprivileged of the general population. Therefore, in spite of favorable reports to the contrary, extreme caution is indicated lest grandparents be sent home to infect their grandchildren where in the past parents have often carelessly been permitted to infect their children. This thought should apply even to the third group of patients with negative sputum findings of seeming reliability.

Surgical resection of the most dangerously involved parts of the lung is now employed with impunity even in elderly patients, but as a group, they are the least suitable for this form of therapy because of the high incidence of complicating factors which impair respiratory capacity and cardiac function.

The social, economic, and emotional problems of old age tuberculosis even over-shadow the medical difficulties. The senile patient is lonely and wretched; often he has neither family nor friends; if he is widowed, his children—themselves beset by poverty—may not be able and at times are not willing to add to their burden by the small and larger ministrations by which they could show their affection and devotion. The old patient is frightened and helpless and his reliance on social and welfare agencies is complete.

Since chemotherapy has come to the fore, the idea of home care for the tuberculous has received widespread attention. Unquestionably, the period of hospitalization can now be shortened and treatment continued at home, but home therapy without an initial stay in a hospital or sanatorium cannot be recommended. It is bound to fail in many aspects of diagnosis

and therapy and deprives the patient of the indispensable education in the meaning and demands of his disease by having contact with other patients and being in the specifically created atmosphere of a tuberculosis hospital. When the time for discharge and home care approaches, the patient's social and economic situation often makes it hard or impossible for physicians and social workers to adjust the humane interpretation of the word "home" to what in reality is awaiting the patient. A flat on the top floor of a cheap walkup rooming house is not home, nor does a room for a whole family in slum quarters deserve that name. In brief, an address cannot be assumed to be a home before a thorough investigation has been made which appraises the home situation on the basis of high standards. Supervised rest and quiet, cleanliness, and comfort are still the mainstay in the treatment of tuberculosis. As yet proof has not been offered that the old methods can be replaced simply by the free provision of antibiotics by the community, even assuming that the drugs are taken as they were prescribed.

The closing of tuberculosis hospitals and sanatoria in reliance on modern chemotherapy is premature. They should serve as the desperately needed homes for homeless aged tuberculous patients where they can enjoy a secure dignified and happy existence. Institutional care for tuberculosis in the sense of this altered interpretation of the term will develop more and more into a crying need as the disease increasingly becomes a geriatric problem. (Bloch, R. G., Tuberculosis - A Disease of Old Age: Arch. Int. Med., 101: 1057-1064, June 1958)

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Localized Pulmonary Emphysema of Infancy

Localized pulmonary emphysema of infancy is now being recognized with increasing frequency. It presents characteristically as hypertrophic pulmonary emphysema localized to one lobe of the lung with infection—if present—having no relationship to the emphysematous process. An emergency situation may arise from the rapidly progressing lobar distention that may terminate fatally without prompt and proper management. Reports of successful treatment by resection of the emphysematous lobe have made this a condition of practical concern to both pediatricians and surgeons.

A variety of names have been given to the condition including infantile lobar emphysema, tension emphysema in infants, progressive infantile emphysema, localized hypertrophic emphysema, lobar emphysema in infants and children, lobar obstructive emphysema in infancy, and congenital lobar emphysema. In many cases, the etiology remains obscure, whereas in others it has been attributed to maldevelopment of the bronchial cartilages or redundant folds of bronchial mucosa. It has been found in association with defects of the mediastinum. In several instances, it has been

associated with bronchial compression by a ductus arteriosus or by pressure from an abnormal vessel. Despite uncertainties concerning its etiology and pathogenesis, the condition does present findings sufficiently characteristic to warrant its consideration as a distinct entity of infancy. It should not be confused with conditions presenting as areas of localized pulmonary emphysema secondary to partial bronchial obstruction associated with infection, atelectasis, tumors, strictures, or aspirated material. Localized emphysema of infancy also needs to be differentiated from lung cysts, atelectasis with compensatory emphysema, pulmonary agenesis, pneumatocele, diaphragmatic hernia, and pneumothorax.

Nothing of significance was noted in the prenatal or birth history of the 40 cases reviewed. Premature births occurred in 4% of the infants with localized pulmonary emphysema. This figure is not remarkable because it falls within the normal incidence of prematurity seen in an average large obstetrical service. Ehrenhaft has suggested that vigorous resuscitation of the newborn might be a factor in the genesis of this condition. Of the 40 cases reviewed by the authors, 17 had no comment concerning resuscitation. In the remaining cases, 22% were definitely stated to have undergone resuscitative measures. Although this figure is higher than one might normally expect, it is difficult to evaluate because there were no detailed descriptions as to the nature and extent of the measures employed.

Sex incidence was significant in that the ratio of male to female infants was 2 to 1. In the present group there were 3 males and 1 female. In the literature, there were 21 male infants, 11 females, and 4 whose sex was not stated.

The predominant symptoms were those associated with progressive respiratory embarrassment. Dyspnea was mentioned in 33 of the 40 cases. Twenty-five infants were noted to have had cyanosis which was often intermittent. In 14 cases, a wheeze had been heard. Feeding difficulty and cough were described in 9 and 8 cases, respectively. Only 6 infants had respiratory infections; these were generally mild and appeared to have had no direct relationship to the emphysematous process. The average time of onset of symptoms was at the age of 25 days. In 17 infants, symptoms began during the first week of life. In the remaining ones, respiratory difficulty became apparent by 2 months of age except for 3 in whom the symptoms began between the second and sixth months.

The usual physical findings were those of respiratory distress; the severity was related directly to the degree of emphysematous lobar distention. The physical sign noted most frequently was mediastinal shift. The location of the apical heartbeat was found to be the most reliable method for determining the degree of mediastinal deviation in infants. Inspiratory retraction, decreased breath sounds, and hyper-resonance over the involved side of the chest were mentioned in slightly less than half of the 40 cases. Cyanosis and dyspnea were stated to be present in approximately

one-third of the cases. In many others, continuous oxygen administration may have masked these signs. Interestingly enough, only 8 infants were noted to have wheeze during hospital admission.

In most instances a detailed history and thorough physical examination with a frontal and lateral chest x-ray will permit a correct diagnosis of localized pulmonary emphysema. Needle aspiration or insertion of an intercostal tube has been performed to relieve the intrathoracic air tension. The authors believe that, unless one is certain of a diagnosis of pneumothorax, these procedures are fraught with considerable danger. In the cases reviewed in which localized pulmonary emphysema was treated by needle aspiration or insertion of an intercostal tube, the results were uniformly bad leading to rapid deterioration and often death.

Progressive localized pulmonary emphysema is best treated by excisional surgery. Any patient suspected of having this disease with unimproved or progressive respiratory distress should have thoracotomy and excision of the emphysematous lobe. Occasionally, thoracotomy may be necessary to establish the correct diagnosis. Thirty-five of the 40 infants had definitive pulmonary resection. The age at the time of surgery varied from 6 weeks to 4 years with more than three-fourths of the patients under 6 months of age. The average age at the time of operation was 6.3 months. In more than half of the cases, the operation would seem to have been elective in nature and to have been performed because of persistent or slowly progressive symptoms. Ten other infants were operated upon as urgent respiratory problems meaning that the operation was necessary within several days after admission to the hospital due to progressive respiratory distress. Three cases were operated upon as emergency procedures within several hours after hospital admission because of their critical respiratory status. (Jewett, T. C. Jr., Adler, R. H., Localized Pulmonary Emphysema of Infancy: Surgery, 43: 926-932, June 1958)

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Acetazolamide (Diamox) Therapy in Chronic Glaucoma

Since 1954, when acetazolamide (Diamox) became available as an additional therapeutic agent for the management of glaucoma, several reports dealing with long-term acetazolamide therapy in chronic glaucoma have been published. A variety of hypotheses have been advanced concerning the mode of action of this drug. At the present time, all that can be said with certainty is that acetazolamide is a specific carbonic anhydrase inhibitor, that it lowers intraocular pressure of human and animal eyes, and that it does so by a partial inhibition of aqueous humor formation. The mechanism of action of this drug as well as its exact locus of action still remains to be demonstrated. Because carbonic anhydrase is known to be present

in lens epithelium, ciliary body, and retina, it became of immediate interest to study two main problems: (a) Does long-term administration of acetazolamide interfere with the metabolic processes of ocular tissues? (b) Will the prolonged administration of acetazolamide irreversibly suppress the process of aqueous formation, i.e., produce a "biochemical cyclodiathermy"?

This report deals with two general aspects: (1) the effect of 2 or 3 years of continuous acetazolamide and miotic therapy on the aqueous dynamics of glaucomatous eyes, and (2) a critical evaluation of the clinical status of patients maintained on long-term acetazolamide and miotic therapy. Of 24 patients with chronic open-angle glaucoma uncontrolled by maximal medical therapy, 21 (87%) had controlled intraocular pressures over a 3-year period of acetazolamide (Diamox) and miotic therapy. The average reduction in pressure was 12 mm. Hg. and the lowered pressure was primarily due to a reduction of aqueous flow (average 60%) as measured by tonography.

The over all percentage of failures in this series of clinic patients was 13 to 21%; 6 of 28 eyes (21%) revealed progressive visual field loss in spite of controlled intraocular pressure.

The exact site and mode of action of acetazolamide still remains to be established, but there is no evidence that long-term acetazolamide therapy irreversibly suppresses the formation of aqueous humor. Following discontinuation of long-term acetazolamide therapy, the intraocular pressure rose to essentially pretherapy levels. The longest follow-up of continuous acetazolamide therapy in this series was 38 months.

Acetazolamide appears to be a relatively safe drug. Mild parathesias occurred in at least 50% of patients. Moderate or severe side effects occurred, but were rare in the authors' experience. There were no significant ocular complications. Vision and visual fields were maintained in many eyes where the prognosis would have been poor without the addition of acetazolamide therapy.

Until longer follow-up studies are available, it is advisable to defer the use of long-term acetazolamide therapy until the various local therapeutic agents have proven inadequate. It should than be used as an adjunct to other therapeutic measures and not as a substitute.

That this study deals solely with chronic simple glaucoma cannot be overemphasized. Except during the immediate preoperative period, acetazolamide therapy has no place in the management of acute congestive or closed-angle glaucoma. (deCarvalho, C.A., Lawrence, C., Stone, H.H., Acetazolamide (Diamox) Therapy in Chronic Glaucoma - A Three-Year Follow Up Study: Arch. Ophth., 59: 840-848, June 1958)

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Surgery of Portal Cirrhosis of the Liver

The surgery of portal cirrhosis of the liver consists in treatment of the secondary effects of this disease rather than a direct surgical attack on the primary condition in the liver. This is a discouraging aspect because surgery does not improve the over all picture of the primary cirrhotic disorder apart from protecting the liver from repeated insults due to recurring esophageal hemorrhages and the nutritional disturbance associated with ascites. Nevertheless, after 13 years of experience with this type of surgery, there can be no question that life has been prolonged in many instances and that the majority of patients have been rehabilitated with a marked reduction in morbidity.

Generally, it is agreed that the chief indication for surgical therapy in portal cirrhosis of the liver is prevention of hemorrhage from esophageal varices—a common source of upper gastrointestinal bleeding and frequently the cause of death in the untreated or medically treated patient. The seriousness of esophageal hemorrhage in patients with cirrhosis of the liver has been recognized for many years. Patek, Nachlas, and Shull have each reported from three different medical centers that when medical measures alone were used, the mortality rate in patients with cirrhosis of the liver after the first hemorrhage from esophageal varices varied from 30 to 50% during the first year. The major causes of death were (1) exsanguinating hemorrhage, and (2) liver failure, in many cases precipitated by esophageal bleeding.

The second indication for surgical therapy in this group of patients is relief of ascites uncontrollable by medical measures. Unfortunately, it is only the occasional patient with marked ascites who can be helped by surgical measures. Remarkable improvement can be obtained in many of these patients by utilization of medical measures, including an adequate diet, low sodium intake, use of diuretics and, if necessary, intravenous administration of human serum albumin. Many patients with bleeding esophageal varices who also have slight to moderate ascites are relieved of their ascites by the construction of some type of anastomosis between the portal venous system and the systemic venous system. Results in a few other patients with uncontrollable ascites and without esophageal varices have been even more spectacular. This is a select group of patients with ascites which does not respond to medical measures despite a relatively normal level of serum albumin, above 3 gm. %.

Any discussion of the surgery of cirrhosis of the liver would be incomplete without considering the emergency treatment of a patient with exsanguinating hemorrhage from esophageal varices because of the high mortality rate in these patients when treated by conservative measures. Statistics collected at the Massachusetts General Hospital in the 5-year period from 1946 to 1950 inclusive reveal an appalling mortality rate in cirrhotic patients with acute, severe esophageal bleeding.

Patients with severe exsanguinating esophageal hemorrhage have been treated as surgical emergencies. The following procedure in these cases has been carried out: First, cardioesophageal tamponage is instituted by use of an intragastric balloon. By this means, it is usually possible to stop bleeding from esophageal varices in a few minutes by applying a 2-pound weight to the end of the balloon tube after the balloon has been inserted into the stomach and inflated. It is recommended that when this has been accomplished, the patient's blood volume be restored by repeated blood transfusion, the operating room be prepared, and in a matter of a few hours, the patient—unless in impending liver failure—be taken to the operating room and the esophageal varices sutured through a transthoracic transesophageal exposure. It is recommended that the operative procedure be carried out in this manner as soon as possible rather than waiting to see if bleeding will recommence when the tube is removed in 24 or 48 hours, which not infrequently occurs; when it does, the patient is usually in a much worse condition to withstand surgery of this magnitude.

It is recommended, therefore, that if cardioesophageal tamponage with a balloon tube is necessary to save a patient from exsanguinating hemorrhage, an emergency operation to suture the esophageal varices should be performed without delay. Fortunately, not all patients with bleeding esophageal varices hemorrhage in this manner so that it is not always necessary to carry out tamponage or emergency surgery. The decision as to which patients should have their esophageal varices sutured should be made by selecting only those in whom it has been necessary to institute balloon tamponage to control the esophageal hemorrhage.

This is not a definitive procedure and for that reason should be considered only the first stage of a two-stage operative program. Fortunately, it controls the bleeding in the majority of patients for a period of 6 weeks to 2 months, thereby permitting more thorough preparation of the patient for the larger surgical procedure of constructing some type of portacaval shunt.

The most effective definitive treatment of bleeding esophageal varices secondary to cirrhosis of the liver that has been developed is the construction of either a splenorenal or a direct portacaval anastomosis. The results to date with this method of surgical therapy have been extremely encouraging. The life of the cirrhotic patients has been prolonged and the incidence of bleeding greatly reduced. In a few patients with uncontrollable ascites, despite their ability to maintain a normal serum albumin level, the construction of a splenorenal shunt has produced spectacular results in the relief of ascites.

It should be emphasized that the success of this type of surgery in many of these critically ill patients demands the closest cooperation of the internist, the surgeon, and the anesthesiologist. (Linton, R. R., The Surgery of Portal Cirrhosis of the Liver: Am. J. Med., XXIV: 941-947, June 1958)

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Surgical Parotitis

Surgical parotitis is an acute inflammation of the parotid gland, either unilateral or bilateral, occurring most frequently after surgical procedures. Although it is an infrequent and unexpected complication, it is quite alarming when it does occur. In the past, it has carried a high mortality rate and has been regarded in the literature as a grave prognostic sign.

It has long been known that patients developing parotitis were usually seriously ill, in poor general condition, dehydrated, undernourished, and with poor oral hygiene. A dry mouth and a decreased salivary secretion seems to be the principal precursor to the onset of the disease. The highest incidence has occurred after abdominal or genitourinary tract surgery.

The infection is almost always caused by the Staphylococcus aureus organism. At the turn of the century, experimental laboratory work was divided in theory as to whether the infection was a hematogenous invasion of the gland or an ascending infection from the mouth by way of Stensen's duct. The latter theory is now more generally accepted. Frequently, the prolonged trauma of pressure on the gland by the anesthetist has been suggested as contributory to the onset of the infection.

The diagnosis is easily made by finding an enlarged tender gland. The onset is usually 4 to 6 days after operation and by far the majority of cases appear within the first 2 weeks following surgery. Occasionally, the first symptom is pain in the temporomandibular joint, but more frequently, there is localized pain and swelling in the gland. A febrile response occurs within 24 hours with temperatures of 102 and 103° F. along with an acute elevation in the leukocyte count. Mortality rates have always been high, varying from 30 to 60% in most series reported. Despite these high figures, parotitis has rarely been considered the primary cause of death; rather, it has been looked upon as indicative of a poor prognosis in those already critically ill.

Early treatment was aimed at stimulating salivary secretion by use of mouth washes, chewing gum, and hard lemon candies, along with warm or cold compresses. Determination of abscess formation by fluctuation is difficult because the parotid gland is covered by an extremely dense and unyielding fascia. Incision and drainage, therefore, have often been delayed. The resulting scar of the face and fear of injury to the facial nerve have been other reasons given for the delay of surgery.

All of these patients but one were acutely ill after extensive intra-abdominal surgery and all were on antibiotic therapy when the parotitis developed. Four cases in which organisms could be cultured were positive for antibiotic-resistant S. aureus. All patients were treated with x-ray as an emergency procedure within the first 12 hours after the onset of symptoms and immediately upon making the diagnosis. The dosage of x-ray was 75 r to gland tissue daily. One patient responded to x-ray therapy only within 48 hours.

Within 48 to 72 hours, when it appeared clinically that the swelling and pain were progressing despite x-ray therapy, six patients were subjected to decompression in the operating room under local 0.5% procaine anesthesia. A hockey-stick incision was started just anterior to the ear and extended 1 inch to a point just below the angle of the mandible and then forward below and parallel to the mandible 1 to 1-1/2 inches farther. The parotid capsule was then split a distance of 1 to 2 inches without incising the gland itself. The authors were impressed by the marked thickening of the parotid capsule which measured 2 to 3 mm. in each instance. No pus was obtained by needle aspiration in any case. The wound was loosely packed with iodoform gauze, and warm compresses were applied for 2 to 3 days.

Relief from pain was almost immediate in each case and marked reduction of swelling was noticed with the first 3 to 4 hours. Recovery occurred in each case during the next 3 to 4 days. There was no evidence of abscess formation in any case. Postoperative scarring was minimal. No x-ray therapy was given after the surgical procedure. One death occurred in this series; that was on the 18th postoperative day of resistant Staphylococcus infection and septicemia. The parotitis had subsided in the meantims. (Gilchrist, R. K., McAndrew, J. R., Surgical Parotitis: Arch. Surg., 76: 863-865, June 1958)

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Practical Aspects of a Hearing Conservation Program

The health problems created by intense noise are becoming increasingly more acute. In addition to the danger of blast and instantaneous excessive noise, continuous and intermittent exposure to loud noises over a period of time may result in hearing loss. This loss may be temporary and recovery may ensue or it may be permanent because of injury of the inner ear. Susceptibility to hearing loss due to noise varies greatly among different individuals; some persons can tolerate much more than others without inner ear damage. Loss usually occurs first in the higher-pitched tones above 4000 cps. Those in whom the loss is centered in this high range suffer quite extensive impairment before the speech range (300 to 3000 cps) is appreciably affected, and long before they become aware of a change in auditory acuity.

There is no clear-cut lower level established above which noise is definitely hazardous to health. Factors, such as frequency, nature of the noise (intermittent or continuous), and the length of exposure greatly influence the level of permissible exposure. A hearing conservation program should be undertaken when the noise level exceeds 85 to 90 db. as measured

on the "B" weighting network on sound-level meter; this coincides in general with conditions that exist when it is difficult to hear a loud spoken voice at a distance of one foot.

Noise measurements and analyses are essential in order to evaluate the extent and nature of the hazard, to determine the need for establishing preventive measures, and to evaluate the effectiveness of a hearing conservation program.

To accomplish ideal control of increased noise output, various approaches are used, examples of which are attenuation of noise at its source by engineering design of machines, substitution of a less noisy operation for a noisy one, isolation to a remote area, acoustical treatment of rooms, resilient mountings, and surrounding the noise source with an enclosure.

Audiograms should be taken in sound treated rooms, or the equivalent, with an ambient noise level no greater than approximately 45 db. The recommended test frequencies are: 500, 1000, 2000, 3000, 4000, and 6000 cps. Hearing acuity is best evaluated on an individual basis. Audiograms should be taken as a part of all preplacement of preemployment physical examinations and routine periodic or recheck audiograms should be made on all personnel working in high-intensity noise areas.

Three general personal protective methods of occluding the ear with devices that attenuate airborne noise are: Insert types—plugs inserted into the ear canal, cushion or doughnut types—objects which cover at least the entrance to the ear canal and often the entire outer ear, and helmet types—fitted coverings for the major area of the head. In some cases, adequate protection may require the combination of these techniques. The occupational medical doctor and his staff should be responsible for the distribution and proper fitting of protective ear devices. Every effort should be made to assist personnel in their understanding and acceptance of ear protection. (Shone, L. B., Captain MC USN, Practical Aspects of a Hearing Conservation Program: Arch. Indust. Health, 17: 610-613, June 1958) (Occ Med-DispDiv, BuMed)

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Policy

The U. S. Navy Medical News Letter, is basically an official Medical Department publication inviting the attention of officers of the Medical Department of the Regular Navy and Naval Reserve to timely up-to-date items of official and professional interest relative to medicine, dentistry, and allied sciences. The amount of information used is only that necessary to inform adequately officers of the Medical Department of the existence and source of such information. The items used are neither intended to be, nor are they, susceptible to use by any officer as a substitute for any item or article in its original form. All readers of the News Letter are urged to obtain the original of those items of particular interest to the individual.

From the Note Book

1. Rear Admiral B. E. Bradley MC USN represented the Navy Medical Department as military member at the House of Delegates Meeting of the American Medical Association held in San Francisco, Calif., June 23-26, 1958. (TIO, BuMed)

2. Rear Admiral E. C. Kenney MC USN represented the Surgeon General at the Twelfth Naval District Symposium on Medical Problems of Modern Warfare and Civil Disaster in San Francisco, Calif., on June 19 and 20, 1958; and at the Sixth Annual National Medical Civil Defense Conference on June 21, 1958.

3. Captain W. M. Silliphant MC USN, Director of the Armed Forces Institute of Pathology, participated in the Joint Meeting of the Canadian Association of Pathologists and the Atlantic Provinces Association of Pathologists held in Halifax, Nova Scotia, 20-21 June 1958. (A. F. I. P.)

4. National Bureau of Standards Handbook 66, was prepared under American Standards Association procedures. As industrial use of radioactive materials, x-rays, and particle accelerators increases, it is essential that adequate precautions be taken to protect the user and the public against excessive exposure to radiation. This Handbook has been composed to serve as a guide toward safe design, manufacture, installation, use, maintenance, and disposal of beta-ray sealed sources for industrial applications. (N. B. S.)

5. "For the Nation's Health" is a 16 mm. filmograph, color, sound, 15 minutes. 1957. This Public Health Service orientation film presents a panoramic view of the activities of the principal health agency of the Federal Government. Combining photographs and motion picture film, it shows the growth of the Public Health Service from its inception in 1798, with limited care of sick and stranded merchant seamen, to its farflung programs today in hospital and medical care, in medical and biological research, and in public health. (P. H. S., H. E. W.)

6. More than 4500 Navy personnel have participated in the Navy's two Antarctic operations, Deep Freeze I and II. Through their efforts, an area the size of the United States and Western Europe combined has been explored, much of it never before seen by man. (Research Reviews, June 1958)

7. Resection of entire lobes of the liver, while not a new procedure, was rarely accomplished until the past 15 years. Right hepatic lobectomy is still so unusual that only a score of patients have been reported as surviving the procedure. This article reports 3 additional patients with benign

conditions requiring hepatic lobectomy, 2 left and 1 right, with excellent results. (Ann. Surg., June 1958; R.C. Clay, M.D., G.G. Finney, M.D.)

8. The authors report their experience with 12 patients with tricuspid stenosis. Five cases are presented in detail to emphasize the variable manifestations of the lesion. The clinical data of the 12 patients are discussed in relation to the hemodynamic data and with particular reference to the symptoms and signs which are considered of aid in the diagnosis. (Am. J. Med., June 1958; T. Killip III, M.D., D.S. Lukas, M.D.)

9. A study was made of 29 patients proved to have sarcoidosis in a 29-month period. The unusually high incidence rate found may reflect partially the inclusion of a number of asymptomatic patients and suggests that the disease is more prevalent than is commonly recognized. A predilection for sarcoidosis apparently exists in both white and Negro patients who have lived in the southeastern part of the U.S. The incidence rate for Negroes in this series was 12 times that of white persons. (Arch. Int. Med., June 1958; Major R.H. Ferguson (MC) and Captain J. Paris (MC) USAF)

10. An operation for the correction of developmental deformities of the anterior chest wall is described in detail. The procedure involves an extensive dissection and mobilization of the sternum and the adjacent anterior chest wall and no external prosthesis or splint is employed. (J. Thoracic Surg., June 1958; R.A. Daniel Jr., M.D.)

11. Cor pulmonale is defined as right ventricular hypertrophy due to a disordered pulmonary circulation regardless of the cause. Pulmonary heart disease, emphysema heart, and pulmonary hypertensive heart disease are types of cor pulmonale. Each refers to right ventricular hypertrophy of more or less specific origin. Brief notes on diagnosis and treatment are presented. (Dis. Chest, June 1958; I.C. Brill, M.D.)

12. In this study, the authors used a simplified classification of peripheral arterial occlusive disease and discussed ulceration, claudication, age, and diabetes as they affected prognosis. (Arch. Surg., June 1958; H.J. Robb, M.D., et al)

13. This report represents a review of the surgical experience obtained from 403 pulmonary resections performed on 338 patients during a 5-year period. The emphasis in this study has been on important surgical complications, their management, and the problems peculiar to the mentally ill. (Surgery, June 1958; A. Mowlem, M.D., et al.)

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Voluntary Retirement

Retirement after 20 or more years of service has been authorized since 1955, and a number of Medical Department officers have been granted this early retirement. It is felt that the availability of early retirement is a distinct addition to the attractiveness of a Navy career.

While general information on voluntary retirement appears to be widely distributed, letters and comments received indicate that some of the details are less widely known. The specific criteria prescribed by the Secretary of the Navy as meriting favorable consideration for early retirement are stated in SecNav Inst. 1811.3A 0f 10 September 1955, and anyone thinking of making such a request should be fully acquainted with this instruction as well as BuPers Inst. 1811.1A of 19 July 1957.

Among the six criteria listed is that of five years' service in grade for captains as well as 20 years' total service. Other of the listed criteria may be applicable to individual cases. Requests are considered on a basis of the over all needs of the Service and the merits of the individual case.

Requests should be submitted at least three months and not more than six months ahead of the desired date, and the preretirement physical must be reported from one to three months in advance. BuPers requires that officers starting a new tour of duty complete at least one year at the new station before voluntary retirement is effected.

Obviously, an unexpected request for retirement creates problems in connection with a relief, and in some instances insufficient time has been allowed in which to arrange for a relief. Consequently, it is most desirable that BuMed be informed of prospective retirement plans as far as possible in advance of the prescribed three months lead time to insure that the desired retirement date can be met.

The Bureau is in no sense urging officers to consider early retirement. This note is simply to urge those who may be thinking of early retirement to become familiar with the requirements and proper procedure as detailed in SecNav and BuPers Instructions. (PersDiv, BuMed)

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Postgraduate Short Courses for Naval Medical Department Officers - Sponsored by the U.S. Army

The following postgraduate short courses will be given during fiscal year 1959 as indicated below. Eligible officers are those who meet the criteria prescribed by BuMed Instruction 1520.8 of 6 February 1956.

Eligible and interested officers should forward requests via official channels, addressed to the Chief of the Bureau of Medicine and Surgery. Requests for attendance must be received in Bureau of Medicine and Surgery

at least 6 weeks prior to commencement of the course requested. Travel and per diem orders chargeable against Bureau funds will be authorized those approved for attendance.

Course	Location	Dates
Management of Mass Casualties	Walter Reed Army Institute of Research, Walter Reed Army Medical Center	15-20 Sep 1958
	Army Medical Service School, Brooke Army Medical Center	28 Jul-1 Aug 1958 22-26 Sep 1958 27 Apr-1 May 1959 15-19 Jun 1959
Eleventh Annual Symposium on Pulmonary Diseases	Fitzsimons Army Hospital	8-12 Sep 1958 (ProfDiv, BuMed)

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Postgraduate Short Courses for Naval Medical and Dental Officers - Sponsored by the U. S. Army and the Armed Forces Institute of Pathology

The following postgraduate short courses will be given during fiscal year 1959 as indicated below. Eligible officers are those who meet the criteria prescribed by BuMed Instruction 1520.8 of 6 February 1956.

Eligible and interested officers should forward requests via official channels, addressed to the Chief of the Bureau of Medicine and Surgery. Requests for attendance must be received in BuMed at least 6 weeks prior to commendement of the course requested. Travel and per diem orders chargeable against Bureau funds will be authorized those approved for attendance.

Course	Location	Dates	Corps Eligible
Forensic Pathology	Armed Forces Institute of Pathology	6-11 Oct 1958	MC
Application of Histochemistry to Pathology	Armed Forces Institute of Pathology	27-31 Oct 1958	MC
Ophthalmic Pathology	Armed Forces Institute of Pathology	9-13 Mar 1959	MC

Course	Location	Dates	Corps Eligible
Pathology of the Oral Regions	Armed Forces Institute of Pathology	23-27 Mar 1959	MC, DC
Cardiovascular Pathology Seminar	Armed Forces Institute of Pathology	6-10 Apr 1959 (ProfDiv, BuMed)	MC

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Vacancies in First Year Residencies in Specialties of Allergy and Otolaryngology to Commence in 1958

Two vacancies now exist for first year level residency training, one vacancy in each of the specialties of Allergy and Otolaryngology, at the U. S. Naval Hospital, San Diego, Calif., to commence at the earliest possible date or during late summer or fall 1958. Applicants for training in Allergy should have had at least one or preferably two years of training in Internal Medicine.

Applications should be made by an official letter addressed to the Bureau of Medicine and Surgery and forwarded via the chain of command, in accordance with BuMed Instruction 1520.10 of 11 February 1957. Applications must contain the Service agreement to serve one year for each year of training received. See enclosure (1) to the above mentioned instruction. Approvals of Reserve officers will be contingent on applying for, and accepting, a commission in the Medical Corps of the Regular Navy, BuPers Instruction 1120.3E applies. (ProfDiv, BuMed)

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Change of Address

Please forward requests for change of address for the News Letter to: Commanding Officer, U. S. Naval Medical School, National Naval Medical Center, Bethesda 14, Md., giving full name, rank, corps, and old and new addresses.

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Use of funds for printing this publication has been approved by the Director of the Bureau of the Budget 19 June 1958.

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Recent Research Reports

Naval Dental Research Facility, NTC, Bainbridge, Md.

1. Microscopic Study of Saliva Sediment. NM 75 01 26.06, 1 March 1958.
2. Survey of Dental Health of the Naval Recruit. II Survey of Dental Treatment. NM 75 01 26.04.03, 1 May 1958
3. Electrophoresis of Saliva. II Reproducibility, NM 75 01 26.05.02, 2 May 1958.

Naval Medical Research Institute, NNMC, Bethesda, Md.

1. Effect of Exposure Geometry and Beam Spectrum on Depth-Dose Patterns for Penetrating Ionizing Radiation in Large Mammals and Man. NM 62 02 00 .01.02, 26 November 1957.
2. Development of Trypanosoma Lewisi in the Heterologous Mouse Host. NM 52 02 00.01.01, 23 December 1957.
3. The Dissimilation of Carbohydrates by Shigella Flexneri 3. NM 52 04 00 .02.04, 30 December 1957.
4. Hexitol Utilization by Shigella Flexneri. NM 52 04 00.02.02, 30 December 1957.

Naval Air Development Center, Johnsville, Pa.

1. Erythrocyte Hydration under Positive Acceleration. NM 19 02 12.1, Report No. 1, 7 April 1958.
2. Variation in Duration of Oculogyral Illusions as a Function of the Radius of Turn. Report No. 2, NM 18 01 12.2, 22 May 1958.
3. Relationship between Pain and Tissue Damage Due to Thermal Radiation. Report No. 15, NM 19 01 12.1, 11 June 1958.

Naval Medical Field Research Laboratory, Camp Lejeune, N. C.

1. Hepatic Function Following Flash Burn. 61 01 09.1.8, March 1958.
2. Acute Blood Volume Changes Following Flash Burn. NM 61 01 09.1.9, March 1958.

Naval Medical Research Laboratory, Submarine Base, New London, Conn.

1. Evaluation of the Radarange for Submarine Use. Report No. 293, NM 24 01 20 .04.02, 22 December 1957.
2. Evaluation for Service Use of a Prototype Swimmer's Rescue Suit. NM 21 01 20.01.01, Memorandum Report No. 58-1, 10 March 1958.

(To be continued in an early issue)

DENTAL SECTION

Greetings from the Surgeon General on the 46th Anniversary of the Dental Corps

Rear Admiral B. W. Hogan MC USN, the Surgeon General of the Navy, addressed the following letter to Rear Admiral R. W. Malone DC USN, Assistant Chief for Dentistry and Chief, Dental Division, Bureau of Medicine and Surgery:

"Rear Admiral R. W. Malone (DC) USN
Assistant Chief for Dentistry
and Chief, Dental Division
Bureau of Medicine and Surgery
Navy Department
Washington 25, D. C.

Dear Admiral Malone:

It is a pleasure for me, as Surgeon General, to extend my sincere congratulations to you and to every member of the Dental Corps on this occasion of the forty-sixth anniversary of the founding of the U. S. Navy Dental Corps.

The Dental Corps has been noted for its progressive effectiveness ever since it was established on 22 August 1912 by the provision of the Naval Appropriation Act which authorized the appointment of 'not more than thirty assistant dental surgeons . . . to serve professionally the personnel of the Naval Service.' During the past forty-six years the Navy Dental Corps has been an important member of our Navy health team. The Dental Corps has justly earned its reputation for being a successful organization. The Bureau is especially proud of the initiative shown recently by the Dental Corps in the fields of developing high-speed operative dentistry techniques and in developing a program to train dental personnel to care for mass casualties. Of course, the most important accomplishment of the Dental Corps has been the excellent level of dental care which its members have provided to personnel of our Navy and Marine Corps. I am especially proud of the effective manner in which dental care has been extended to overseas dependents during the past year, as authorized by the Dependents' Medical Care Act.

On this forty-sixth anniversary, I also wish to extend my congratulations to the Dental Technicians, Dental Service Warrant Officers, and Medical Service Corps Officers, whose skill and loyalty have contributed so much to the accomplishments of the Navy Dental Service. I join all of you in looking forward with confidence to many more years of successful achievements.

Anniversary best wishes,

Sincerely,

/s/

B. W. Hogan
Rear Admiral, MC USN
Surgeon General"

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Practical Methods for Good Dental X-Ray Practice

Check the Dental X-Ray Machine. Use only modern, well shielded equipment. Obsolete equipment is likely to be dangerous both electrically and radiologically and should be discarded, particularly old units with open tubes. The modern high kilovoltage machines are preferable. Dental x-ray equipment should be used for dental radiography only—it is not designed for other purposes.

Use the Proper Diaphragm or Cone. The apparatus should be tested to be sure that there is no appreciable leakage radiation emerging through any other part of the tube housing or the diaphragm or cone margins. The proper diaphragm or cone limits the primary beam to a circle of 3 inches diameter at the tip of the cone. If this must be removed for a special purpose, such as examination of the temporo-mandibular joint, the regular cone or diaphragm should be replaced before subsequent conventional use.

Use the Proper Filter. Make certain that the filter is always in place. It should be at least 1.5 mm. of aluminum in machines which operate up to 80 KVP and at least 2.0 mm. of aluminum in machines which operate above 80 KPV.

Measure the Output of the Machine. The radiation output of the machine should be known under the conditions of kilovoltage, filter, and distance used.

Maintain Adequate Radiation Protection for Operating Personnel. All personnel should stay well away from the primary beam. The operator should never hold the dental film, the pointer cone, or tubehead, during exposure. The use of handheld fluoroscopic screens is dangerous and should

be strictly avoided. The timer control cord should be long enough to allow the operator to stand behind an adequate protective barrier. When this cannot be arranged, the operator should stand at least 5 feet away from the tube and well away from the primary beam. Film badges are recommended for monitoring of personnel exposure. They should be of the type designed and processed for this purpose; ordinary dental films with a paper clip or coin are not satisfactory for this purpose.

Check the Radiation Protection of the Room Installation. The safety of adjacent areas from both primary and secondary radiation should be assured with protective barriers and distance as specified in National Committee on Radiation Protection Handbook #60 and applied to the particular installation. It is good practice to rotate the dental chair so that the patient faces away from the room window during exposures, as this directs the primary beam toward an outside wall.

Use Fast Film, Good Exposure, and Processing Technique. The radiation exposure of patient and personnel can be greatly reduced by using high kilovoltage, adequate filters, and modern fast film. The longest target to film distance that is practicable should also be used. The optimum exposure times for these factors should be carefully determined and used regularly. All film processing should be done with exact time and temperature control, using fresh and good solutions. Attention to these points will give the best quality films and will reduce the need for reexaminations with their attendant additional radiation exposure.

Protect the Gonads of the Patient. Whenever possible, direct the primary beam away from the region of the gonads. This can often be achieved by appropriate tilting of the patient's head. Some additional reduction in primary or scattered radiation can be obtained by placing protective material, such as a lead rubber sheet or apron, across the patient's lap. This is particularly useful when multiple exposures are necessary or in special instances, such as children and pregnant women or when the primary beam cannot be directed away from this area.

Consider the Indications for Each Examination Performed. Is is good practice to do a limited examination first. This preliminary survey will often provide all of the necessary information. Additional film exposures can then be decided upon as specifically required. "Routine" extensive surveys and frequent reexaminations should be avoided whenever possible.

Use Special Care with Children and Pregnant Women. The number of exposures to children should be restricted to an absolute minimum from both somatic and genetic considerations. The extra hazard of exposure to pregnant women calls for postponing complete or elaborate procedures as much as practicable during this period. (Chamberlain, R. H., Nelson, R. J., prepared by the American College of Radiology - A Practical Manual on the Medical and Dental Use of X-Rays with Control of Radiation Hazards)

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RESERVE SECTION

Reserve HC Division Participates in Operation Alert

At 0800 hours on 6 May 1958, members of Naval Reserve Hospital Corps Division 4-2 Pittsburgh, Pa., commenced participation in operation "Prep Pitt" which was developed as a simulated training exercise under the nationwide "Operation Alert."

Under the joint sponsorship of the Allegheny County Medical Society and Hospital Council of Western Pennsylvania, the operation was conducted in cooperation with the Medical Education for National Defense Program of the University of Pittsburgh School of Medicine, the Office of Civil Defense, City of Pittsburgh, the Pittsburgh Chapter of the American Red Cross, and the Veterans Administration Hospital, Pittsburgh, where casualties were received and treated.

Designed and planned to demonstrate problems associated with a modest disaster, operation "Prep Pitt" was based on 50 casualties who were survivors of an explosion and fire following a crash of a jet aircraft on the University of Pittsburgh Field House. The "injured" included patients suffering from burns, soft tissue injuries, cavity injuries, and fractures. All were moved to the Veterans Administration Hospital.

After 7 hours continuous drill, the exercise was completed at 1500. For their vigorous and effective performance of tasks assigned, members of Hospital Corps Division 4-2 were commended by both the staff of the Veterans Administration Hospital and officials assigned to umpire the operation.

In addition to evolving a more effective disaster plan and providing excellent training for care of mass casualties, recruiting of new members for the unit has been stimulated.

A salute to LT E. E. Longabaugh MC USNR, Commanding Officer, and the members of 4-2 for their efforts in this laudable undertaking.

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Military Industrial Vision Seminar

The California Optometric Association will conduct a Military Industrial Vision Seminar at the Lafayette Hotel, Broadway and Linden Avenues, Long Beach, Calif., 28 - 29 August 1958.

The Chief of Naval Personnel has authorized retirement point credit to Reserve Medical Department officers for daily attendance providing they register with the authorized military representatives present. Participation in this seminar offers an excellent opportunity for Medical Department officers to be brought up to date in the latest developments of industrial optometry.

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Treatment of Chemical Warfare Casualties

The Medical Department correspondence course, Treatment of Chemical Warfare Casualties, NavPers 10765, is available to Regular and Reserve officers and enlisted personnel of the Medical Department of the Armed Forces as well as officers of the U. S. Public Health Service and allied foreign medical department officers.

Modern chemical warfare began with the use of chlorine gas by the German Army in 1915. This new kind of warfare had a psychological effect. Coupled with misleading propaganda and a lack of knowledge of the specific chemical agents used, it resulted in unfounded fear in the troops and misdiagnoses by medical personnel. As a result of better understanding of the physiological and psychological effects of chemical warfare agents, military personnel are now better equipped to cope with gas attacks than were military personnel in the past.

The purpose of this course is to provide personnel with important self-aid techniques for battle field treatment of chemical warfare injuries and to assist the enrollee in explaining these techniques to individuals who may encounter gas warfare situations. The course also describes methods of detecting and identifying agents, techniques of identifying casualties and noncasualties, and methods of protecting personnel prior to exposure to chemical agents. The physiological and psychological effects of each agent are discussed, together with the recommended treatment for each. In the event of a future chemical warfare situation, personnel who have completed this course will be aware of the important self-aid techniques for battlefield treatment of chemical warfare injuries. Knowledge of the properties and effects of these agents will assist personnel in the prevention of dangerous psychological effects on military personnel.

The course consists of three (3) objective type assignments and is evaluated at nine (9) Naval Reserve promotion and/or nondisability retirement points. Applications for this course should be forwarded on NavPers 992 (Rev 1/57) via applicant's command to Commanding Officer, U. S. Naval Medical School, National Naval Medical Center, Bethesda 14, Md. Make appropriate change in the "To" line in Box J of the application form.
Note: Medical Department Reservists may enroll in more than one MD correspondence course at one time.

PREVENTIVE MEDICINE SECTION

Seat Belts in Actual Crashes

Automobile seat belts have chalked up an amazing record for taking the stress of accident crashes. Cornell University's Automotive Crash Injury research has found that only 11 seat belts involved in 511 accident cases studied failed to function as intended. A total of 712 belts was involved in the 511 accident cases processed.

The researchers observed that, even in the 11 cases of misfunction, the failure to operate as intended could not always be ascribed to structural deficiencies of the belts themselves. Here is a brief description of the types of failure followed by some actual case histories:

Webbing Failure. Among the 11 cases, there were 4 in which the webbing parted. Two of these were clear-cut cases in which the webbing of a relatively new belt failed to stand up under impact. In a third case, the webbing which parted had been in use for 9 years prior to the accident and the failure can probably be ascribed to normal deterioration with age. In the fourth case, both the webbing and the floor attachment were reportedly "ground to pieces" by direct impact when a large tree impacted at some 50 mph penetrated the passenger compartment.

Case: A 1954 Austin sport roadster missed a curve and crashed into a tree 24 inches in diameter. Traveling speed prior to accident was 70 mph and at impact about 50 mph. The point of impact was the right front side just ahead of the door. The accident is classed an "non-survivable" for the right front seat occupant, with or without safety belt. The right front passenger was thrown out when his belt failed. Fatal skull fracture was ascribed to impact with pavement when ejected. The driver suffered lacerations of the scalp and right eyelid (windshield), a bruised chest (steering assembly), and contusions of right elbow, right hip, left ankle, and chin.

Buckle Slippage. Of the two cases where belts slipped through the buckles, one was a case in which the trooper reported that the belt was improperly threaded into the buckle. In the other, a trooper reported that

his own buckle failed to hold when his weight was thrown against it, but did not specify whether it was properly engaged.

Case: The front end of the case car, a 1954 Tudor Ford, struck the side of a 1946 Chevrolet pickup truck. Speed of case car, 40 mph. Speed of truck, 30 mph. Driver states that buckle did not hold when his weight was applied against it, but nevertheless, felt that the belt did afford some protection. Belt was attached at floor. Driver suffered bruises and contusions to right knee which struck dashboard. No passengers.

Anchorage Failure. Two cases were reported, one of which also involved parting of the webbing. In both cases, there was considerable evidence that the anchorages were sheared off by direct impact when obstacles struck penetrated the passenger compartment. Both were cases in which there was some question as to whether the accident was technically a "survivable accident," because the passenger compartments of the cars involved were extensively damaged.

Case: The case car, a 1953 Studebaker two-door sedan, attempted to dodge a 1947 Plymouth two-door sedan which was on the wrong side of the road. Speed of the case car was 55 mph. Speed of other car prior to impact was 55 mph, at impact, 50 mph. The left front half of the Plymouth plowed along the right side and penetrated the rear seat area. When the floor attachment of the right front passenger's belt twisted and broke off, he fell against the glove compartment and toppled out of the car. He suffered deep laceration on right shoulder (glove compartment), minor facial abrasions and contusion of right hand (cause unknown), and contusion of the kidney and hematuria (inside or outside car). The driver suffered a four-inch scalp laceration (windshield) and bruises on chin (upper steering wheel).

Buckled Floor Pan. In one case, although the belt held, the floor pan to which it was attached buckled upward about six inches. This is clearly not a case of belt failure, but does indicate the importance of considering the strength of structures to which belts are to be attached.

Case: The left front third of the case car, a 1950 Plymouth 2-door sedan, struck a tree about one foot in diameter. Car turned over on its left side. Speed prior to, and at, impact was 50 mph. Left front door opened at impact. The driver suffered bump on forehead (steering wheel), minor cuts on face (flying glass), bruised left elbow (door structure), and bruised left ankle (pinched by seat). There were no passengers.

Webbing Slipped out of Anchorages. Two cases, both in the same car, were reported. The anchorages were of the triple-slot variety, where

the belt is threaded in a precise pattern through the series of slots, and held by friction. Because there are numerous cases on record where such attachments have held, but because the threading pattern must be precisely followed to hold properly, it is at least possible that in this case the belts' ends were improperly threaded. This appears especially likely since the two cases of failure were in the same car, and the two belts were probably threaded by the same person.

Case: The case car, a 1955 Ford tudor sedan, traveling at 79 mph, went out of control on the right shoulder, then swerved back across the highway into oncoming lane. A 1956 Oldsmobile, two-door sedan, traveling at 55 mph ran into the right fender of the case car. Both cars spun around and left the highway. Doors of case car opened and right front passenger was ejected and killed from injuries sustained outside the car (broken neck, unspecified head injuries, fractured left femur). The driver suffered a fractured right arm and lacerated right hand (cause unknown).

Special Circumstances. In one case, where design was such that the outer strap of the belt was attached to the door, some slack was caught between the door and the seat by mistake. When the door came open, the belt no longer functioned as a seat belt, but became in effect merely a door strap. Although the belt remained structurally intact, the occupant was readily ejected. (Traffic Safety, 52: 10-12, June 1958)

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Possible Transmission of Poliomyelitis by Domestic Pets

Although numerous animals, including dogs, cats, and even moles, have been named as possible reservoirs of poliomyelitis virus, confirmatory evidence from laboratories has always been lacking. Possible avian reservoirs were suggested when a time relationship was observed between outbreaks of fowl paralysis and the human poliomyelitis epidemic in South Africa in 1948, but no association was proved.

An incident is described by Dr. R.G. Sommerville and his associates (The Lancet, March 8, 1958) in which there seems little reason to doubt that a budgerigar (Australian parakeet) became infected with poliovirus type 1 and excreted this virus for at least 3 weeks. Again, attention is drawn to the possibility of transmission of poliomyelitis by domestic pets.

A budgerigar recovering from paralysis of its legs bit a boy on the lip, holding on with its beak until it was removed by the boy's mother. Another bird which had been kept in the same cage had died from a similar

attack of paralysis a few days earlier. A week after the bite, the child developed signs of bulbar poliomyelitis and subsequently died. Type 1 poliovirus was recovered from his stool.

A possible association between the bird's bite and the boy's illness led to an examination of the bird's droppings which were found to contain type 1 poliovirus. A second sample of droppings and of the bird's intestinal contents, recovered after sacrifice of the budgerigar 3 weeks later, yielded the same findings.

The viruses were identified by neutralization tests in HeLa cells using high-titer antiserum prepared in monkeys against standard strains of poliovirus types 1, 2, and 3. No difficulty was experienced with the neutralization tests and the viruses behaved like any other strains of poliovirus type 1.

To exclude the possibility that the monkey antiserum to poliovirus type 1 which was used to identify each virus contained additional antibody to an agent other than poliovirus, a rabbit antiserum was prepared against the virus isolated from the intestinal contents of the bird at necropsy. The rabbit antiserum neutralized the standard strain of poliovirus type 1 (Mahoney) maintained in this laboratory and also neutralized each virus isolated from the budgerigar. The antiserum did not neutralize either type 2 or type 3 poliovirus.

A similar case is recorded (Dublin City Health Dept., 1956) involving a household in which a child developed paralytic poliomyelitis (poliovirus type 1 found in feces) and in which 23 canaries had died about the same time. No leg paralysis had been observed in any of the birds, and there was no history that the child had been pecked by the canaries, but had played in the room where they were caged. No laboratory examination was made of excreta from these birds.

Heretofore it has been thought that under natural conditions no animal other than man becomes infected with poliomyelitis. This opens a whole new field for speculation and research in regard to poliomyelitis virus. An explanation of the seasonal appearance of poliomyelitis may come through discovery of a reservoir for the virus during the months of low incidence.

(CommDis, PrevMedDiv, BuMed)

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Ecology of Equine Encephalomyelitis

This article summarizes a series of field investigations which constitute a continuation and geographic extension of previous studies. The data consist of the results of virus isolation and serum neutralization tests on wild bird blood collected in Louisiana, Alabama, New Jersey, and Massachusetts. When the results of studies made on eastern equine encephalomyelitis (EEE) in wild birds in different localities over the course of several

years are summarized, two differing patterns of activity appear to prevail. The first pattern appears to be the progression of the virus through a wild bird population at a normal endemic maintenance rate. This appears to have been the case in Louisiana in 1952, 1953, and 1956, and in Massachusetts in 1953. Also it presumably was the case in New Jersey in 1955 and in Alabama in 1956 as indicated by the residual immunity rates observed early the following year. Under these conditions, it was possible to isolate virus from less than 1% of the birds collected and 13 to 22% of the population were found to possess neutralizing antibody. Very little or no human or horse involvement occurred in any of these areas during the years mentioned. Such a level of activity would seem to favor the continued presence of the virus.

A second pattern of activity of EEE virus in wild birds was observed in two different localities in New Jersey and also in Massachusetts during 1956, and in Alabama in 1957. Also it may be presumed to have occurred in Louisiana in 1955 on the basis of the 54% immunity rate observed in the March 1956 collection. A similar situation appears to have been present in New Jersey in 1953. On these occasions the virus seems to have spread through the wild bird population with explosive speed. Bird species, such as English sparrows and domestic pigeons which are not involved in the endemic sylvan cycle became involved. On several occasions, virus was isolated from as high as 11% of the birds in a day's collection and this activity was seen to result in the immunity of 45 to 54% of the population. Such a level of immunity would seem to jeopardize the continued presence of the virus. The virus also appeared outside its usual geographic limits and on all of the instances mentioned, equines were involved in epidemic proportions. Human cases of EEE occurred in Louisiana in 1955 and in Massachusetts in 1956. EEE epidemics also occurred in ring-necked pheasants in New Jersey in 1953 and 1956, and in Massachusetts in 1956.

Such hyperactivity also seems to have occurred with western equine encephalomyelitis (WEE) virus in Louisiana in 1952, and in New Jersey in 1956. However, the over all rate of activity of WEE in the eastern United States seems to be lower than that of EEE, minimizing the importance of such occurrences proportionately.

The ecologic balances which hold the activity of EEE virus to a level compatible with its survival in appropriate areas are not well understood. However, it seems logical to conclude that its maintenance depends on sufficiently susceptible bird populations of appropriate density and upon a vector population of proper transmitting efficiency and also at an optimum density. These balances may be upset, conceivably by great increases in numbers of either birds or the usual vectors. It is more probable, however, that it is the entrance into the transmission cycle of other highly efficient mosquito species in large numbers which produces an epidemic situation.

Conditions seem to be well established for the maintenance of these viruses in the eastern United States and to be of such longstanding and wide

occurrence that their eradication from nature is probably impossible or impractical. On the other hand, an understanding of the factors responsible for these sudden presumably abnormal bursts of virus activity may enable workers to predict epidemics or to detect them in their incipient stages, and possibly prevent the involvement of man or domestic animals. (Stamm, D. D., V. M. D., Studies on the Ecology of Equine Encephalomyelitis: Am. J. Pub. Health, 48: 328-335, March 1958)

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Fires and Explosions in the Operating Room

Fires and explosions have occurred in conjunction with the use of flammable anesthetics since their introduction. Has the problem of preventing fires and explosions in the operating room gradually become more complex? The answer is definitely "yes" and the reasons are not hard to find. The increased number of operations and the greater length of many of them have multiplied the hours during which fires and explosions might occur. A host of electrically operated pieces of apparatus, such as cauteries, suction pumps, electrosurgical, electrocardiographic, and electroencephalographic units, photographic equipment (movies and television apparatus) have found their way into the operating room. Also the increased number of individuals in the operating room has multiplied the chances for the ignition of flammable mixtures by static sparks. The surgical invasion of all compartments of the body (chest, heart, brain) and the performance of radical surgery therein so often requiring the use of electrosurgical equipment have made impractical the attempt to keep the surgeon and his dangerous equipment at a "safe distance." Do we have a solution to the problem of fire and explosion in the operating room? No, not completely, but progress has been made and much can be done to decrease this hazard.

Flammable mixtures require an ignition source for an explosion to occur. These sources of ignition can be divided into four general groups: (1) direct contact with open flame or hot bodies, (2) sparks from electrical power circuits, (3) electrostatic discharge, and (4) spontaneous combustion.

The personnel should have basic initial instruction and periodic refresher sessions to remind them of the many hazards that exist in the operating room. The desirability for them to realize the importance of the danger zone which exists at the head of the table while flammable anesthetics are being administered cannot be overemphasized. A philosophy of slow, deliberate motion should be established—particularly among anesthesiologists. An electrostatic charge can accumulate only when the rate of generation exceeds the rate of dissipation over the conductive pathway. Slow motion in the hazardous area becomes an important safeguard when contact with the conductive pathway is marginal.

Dangerous fabrics must not be worn. Freshly laundered cotton with an adequate moisture content is essential for electrostatic control. The buildup of electrostatic charge on wool and synthetic cloth is so rapid that these materials are outlawed unless the entire garment is worn in direct contact with the skin which serves as a leakage path for the dissipation of the charge. For example, nylon hose present little hazard once the soles are moistened with perspiration; a petticoat, however, is dangerous because the free hanging skirt becomes charged through motion. Synthetic material is such a good insulator that the charges remain even though the bodice is in contact with skin. There must be personal responsibility for maintenance of a conductive contact with the floor. Conductive sole shoes, conductive booties or slip-ons are effective only when they are properly worn. They must be tested daily because dirt that collects on the bottom may destroy the electrical contact. A testing device and an inverted brush fixed to the floor should be standard equipment at the entrance to every operating room corridor so that faulty contact can be detected and corrected. Because dirt interferes with the electrical contact, personnel must be trained to keep the floors clean. The surgeon who discards suture ends to the floor, the nurse who drops broken glass or fails to clean up blood or pus before it is tracked about, contribute to the hazard. (Nicholson, M. J., Orr, R. B., Fire and Explosion Hazards in the Operating Room: Surg. Clin. N. America, June 1957: Abstracted in J. A. M. A. 167: 52, May 10, 1958)

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Formulas for Newborn Infants

The preparation of baby formulas is a relatively simple task which can be performed by any well trained careful hospital corpsman.

A baby's formula should be as sterile as humanly possible to provide. Bacteriologic examination should be performed at least once a week. The plate counts on random samples of bottled formula should not exceed 25 organisms per milliliter. Generally prescribed formulas of either evaporated milk or sterile powdered milk permit this standard to be maintained without difficulty, if the proper method of terminal heating is observed.

An authoritative guide for formula preparation, Standards and Recommendations for Hospital Care of Newborn Infants, published by the American Academy of Pediatrics, 610 Church Street, Evanston, Ill., is available at $1.50 per copy. (Sanitation Section, PrevMedDiv, BuMed)

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Statement on Prophylaxis of Ophthalmia Neonatorum

On January 25, 1955, the Committee on Fetus and Newborn reviewed the problem of prophylaxis of gonorrheal ophthalmia in the newborn infant with particular reference to the possibility of changing the standing recommendations for instillation of 1% silver nitrate. The review of available information indicated:

1. That no evidence existed that damage to the eyes has followed the use of 1% silver nitrate when used as recommended.
2. That use of various antibiotics (penicillin, erythromycin) instead of silver nitrate did reduce the incidence of nonspecific conjunctivitis.
3. That the antibiotics used in controlled studies apparently were as effective in preventing gonorrheal ophthalmia as was silver nitrate, e.g., no cases were reported in either group.

The Committee was concerned with the possible occurrence of sensitization to locally administered antibiotics, the possible emergence of antibiotic resistant strains of bacteria, and possible difficulties in maintaining stability and potency of antibiotic preparations under hospital storage conditions. Furthermore, the Committee was aware of studies in progress in which no routine prophylaxis was carried out. In view of these considerations, the Committee on Fetus and Newborn did not recommend any change in the existing procedure for the prevention of gonorrheal ophthalmia.

On February 8, 1958, the Committee again reviewed the problem in light of additional information:

1. In a large metropolitan hospital, elimination of eye prophylaxis of any type resulted in the occurrence of 4 cases of gonorrheal ophthalmia within 4 months. Significantly, none of the others had given any evidence of disease and one of the babies developed conjunctivitis after discharge from the hospital. This hospital had had no cases of gonorrheal ophthalmia since the institution of silver nitrate prophylaxis.
2. In a study from Australia, babies receiving no prophylaxis demonstrated as high an incidence of discharging eyes as did babies receiving silver nitrate. Furthermore, babies receiving no prophylaxis demonstrated a higher incidence of pathogens on culture (nongonococcus) than did the babies receiving silver nitrate. The most common organism found in both groups was staphylococcus aureus.
3. Various prophylactic measures were considered in the context of the problem of antibiotic resistant infections in newborn nurseries together with the fact that the organisms commonly found in the eyes of newborn infants have very limited sensitivity to currently available antibiotics.

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On the basis of available evidence, the Committee believes:

1. That gonorrheal ophthalmia still constitutes a definite hazard to the newborn infant.
2. That silver nitrate has amply demonstrated its effectiveness as a prophylaxis of gonorrheal ophthalmia.
3. That the occurrence of nonspecific conjunctivitis does not of itself constitute adequate reason for change.
4. That the routine use of antibiotics may introduce further problems referable to control of infections in new born nurseries.

Therefore, the Committee on Fetus and Newborn recommends that the routine use of 1% silver nitrate for prophylaxis of gonorrheal ophthalmia be continued. (News Letter, American Academy of Pediatrics, Vol. 9, No. 3, March 1958; abstracted in Connecticut Health Bulletin, June 1958)

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